**Original Article**

# Derivation of a Prediction Model for Emergency Department Acute Kidney Injury

**Short title (running head)**

Prediction model for ED-AKI

**Abstract**

**Background and Objective:** Quality management of Acute Kidney Injury (AKI) is dependent on early detection, which is currently deemed to be suboptimal. The aim of this study was to identify combinations of variables associated with AKI and to derive a prediction tool for detecting patients attending the emergency department (ED) or hospital with AKI (ED-AKI).

**Design, setting, participants and measurements:** This retrospective observational study was conducted in the ED of a tertiary university hospital in Wales. Between April and August 2016 20,421 adult patients attended the ED of a University Hospital in Wales and had a serum creatinine measurement. Using an electronic AKI reporting system, 548 incident adult ED-AKI patients were identified and compared to a randomly selected cohort of adult non-AKI ED patients (n=571). A prediction model for AKI was derived and subsequently internally validated using bootstrapping. The primary outcome measure was the number of patients with ED-AKI.

**Results**: In 1119 subjects, 27 variables were evaluated. Four ED-AKI models were generated with C-statistics ranging from 0.800 to 0.765. The simplest and most practical multivariate model (model 3) included eight variables that could all be assessed at ED arrival. A 31-point score was derived where 0 is minimal risk of ED-AKI. The model discrimination was adequate (C-statistic 0.793) and calibration was good (Hosmer & Lomeshow test 27.4). ED-AKI could be ruled out with a score of <2.5 (sensitivity 95%). Internal validation using bootstrapping yielded an optimal Youden index of 0.49 with sensitivity of 80% and specificity of 68%.

**Conclusion**: A risk-stratification model for ED-AKI has been derived and internally validated. The discrimination of this model is objective and adequate. It requires refinement and external validation in more generalisable settings.

**Keywords (MeSH)**

Adults; acute kidney disease; clinical prediction score; emergency department; risk score

## 1. Background

Acute Kidney Injury (AKI) is a global health issue complicating 4-20% of all hospital admissions with an incidence as high as 70% in critically ill patients [1, 2]. It is characterised by an abrupt loss of kidney function that is strongly associated with high mortality and morbidity [3, 4]. In patients attending the University Hospital of Wales as emergencies with AKI, the 30-day mortality is 26% [5]. The majority of these patients are admitted through the hospital front door either through the emergency department (ED) or the acute medical admissions unit.

In 2009 the National Confidential Enquiry into Patient Outcomes and Death (NCEPOP) **in the United Kingdom** reported that the recognition, management and documentation of AKI was gravely suboptimal especially in those patients who died with an AKI [6]. **NCEPOP made the case that management was poor due to a poverty of accurate early diagnosis and early intervention.** The literature is replete with calls for early diagnosis and early intervention of AKI. The National Institute for Health and Care Excellence (NICE) recommends that risk assessment is made at the hospital front door **especially therefore in emergency departments** [7]. Yet there are no validated bespoke risk tools for detecting AKI let alone high-risk AKI in the context of the emergency department.

**It is rare that AKI is immediately life-threatening. As a result it receives little attention from emergency physicians. Yet delays in detection and treatment result in an in-hospital mortality rate of 20 – 40% that is higher than acute stroke and acute myocardial infarction combined [Holmes 2016; NICE 2019]. We have previously shown that more cases of index hospital AKI e-alerts occur in the emergency department (53%) than the rest of the hospital combined and that this is true of all three stages of AKI [Holmes 2016]. Also, 24% cases of AKI deteriorate significantly to a higher stage relative to the incident creatinine measurement and the mortality in this group is 38.8% [Foxwell 2019]. We have also shown that ED-AKI is an independent risk factor for mortality and that in those patients who die with associated AKI, 58% occur within 7 days of admission to hospital. There is little other, if any, specific data on the recognition, detection and treatment of AKI in the ED. However, the NCEPOD report on hospital mortality primarily due to acute renal failure includes EDs [NCEPOD]. It judges that *in pre-admission AKI*, good clinical practice occurred in only 55% cases, assessment of risk factors was poor,**

**subsequent delays in recognition occurred in 43% cases, that a fifth of post-admission AKI was predictable and avoidable and that complications were missed in over 50% cases.**

The introduction of an electronic alert in Wales was an attempt to facilitate early detection of AKI [8-10]. Once an elevated creatinine is identified in the laboratory an e-alert is triggered. However, the e-alert is dependent on an appropriate urea and creatinine blood tests being requested. Further, the evidence is that the introduction of the e-alert makes little difference to outcomes [11].

We hypothesise that information that is easily obtained from adult patients at a triage assessment in an emergency department (ED) may be used to determine the risk of ED-AKI in patients and to guide the request for ED screening tests and early monitoring of bladder volume and urinary output and quality. The purpose of this retrospective, single-centre, observational study in an ED and acute medical unit (AMU) was to identify combinations of add-on tests to estimate the probability of ED-AKI and to produce a model for risk-stratifying such patients. Specifically, we aimed to estimate the accuracy of individual variables at ED clinical presentation for predicting ED-AKI. Secondly, we aimed to derive and internally validate a prediction score for ED-AKI.

## 2. Methods

### 2.1. Ethics and Study design

Ethical approval has been obtained from Cardiff University and Cardiff and Vale UHB to conduct a retrospective, single-centre, study in the ED and AMU of the University Hospital of Wales, Wales, UK. Written consent was not required from all participants as the study was retrospective, anonymized, complied fully with the Declaration of Helsinki and Good Clinical Practice Guidelines and was part of an ongoing quality improvement project.

### 2.2. Setting and Electronic Reporting System

The study was conducted in the ED and AMU of the University Hospital of Wales which receive 300 and 20 new patients respectively each day and which are associated with the Cardiff and Vale Nephrology and Transplant directorate. This

centre is a tertiary referral centre providing all aspects of renal services for patients in South East Wales.

### 2.3. Participants

The study cohort including basic characteristics, data collection, epidemiology, demographics and outcomes have been previously reported [5]. Out of 17,693 patients attending the ED, 548 incident adult ED-AKI patients were identified. A non-AKI control cohort of 600 patients was generated from the 17,145 incident patients attending the ED with no AKI alert by random selection using the random number generating function.

### 2.4. Measurements

Twenty-seven variables collected by research staff are shown in Table 1. They include patient characteristics, medical history, conventional risk factors including diabetes, hypertension, lipid profile, smoking history, family history and investigations which may be available within an emergency department. We did not collect point of care venous blood gas, current medication, laboratory tests, or physiological observations, as these were not available in the administrative repository.

### 2.5 Definitions

The definition of AKI accord with the current 'Kidney Disease: Improving Global Outcomes' (KDIGO) guidelines [8]. Patients for whom the first e-alert was generated from a creatinine value measured in primary care were classified as primary care AKI.

Pre-existing chronic kidney disease (PeCKD) was defined as an eGFR (calculated by modified MDRD formula) <60ml/min/1.73m$^2$ derived from the baseline SCr [12]. 'PeCKD and worsening eGFR' was defined as a decline in eGFR >15% or a decrease in eGFR >5ml/min/1.73m$^2$ (with a baseline eGFR <60ml/min/1.73m$^2$) [13].

### 2.6 Outcomes

The primary outcome measure was the number of cases with ED-AKI. ED-AKI is defined as occurring in the ED prior to discharge or admission.

### 2.7 Statistical Analysis

The unit of analysis was the patient. Statistical analysis was carried out using MedCalc 18.5 – 64-bit for Windows XP/Vista/7/8/10 (MedCalc Software, Ostend, Belgium). Descriptive statistics are presented as medians and interquartile ranges, means and standard deviations or 95% confidence interval (95% CI) and numbers and percentages.

Initially univariate analysis was used to identify variables that may be used in the model. Univariate logistic regression was performed on all 27 variables in order to determine the unadjusted odds ratios. P values ≤0.05 were considered statistically significant.

Models were then devised according to the available data along specific steps of the patient pathway. Models 1 to 3 include data only available at ED triage. Model 4 includes data available at triage and early in the clinical pathway.

Multivariate Cox proportional hazard modelling was used to determine the significance of variables for ED-AKI. Stepwise backward variable selection with significance level <0.05 were considered statistically significant. Several combinations were tested depending on the patient's place in the ED pathway and the availability of data.

For the most practical score a risk score was devised by rounding the raw regression coefficients to the nearest integer. The risk score was then estimated for each patient according to the results of the two diagnostic tests. The coefficients of each of the variables were summed and rounded in order to develop a 31-point risk-score where 0 is lowest risk and 31 is highest. After performing logistic regression where the dependent variable was ED-AKI and the independent variable 'risk score', we re-assessed the calibration of the model.

The performance of the model to predict the risk of ED-AKI was estimated by calibration and discrimination. Calibration was assessed using the Hosmer–Lemeshow goodness-of-fit test. Discrimination was assessed using the area under the receiver operating characteristic curve (AUC, c-statistic). The model accuracy was thus reported as sensitivity and specificity, goodness of fit, and AUC.

The model was validated internally using bootstrapping with 1000 itinerations each with 978 randomly selected samples. This generated an optimal sensitivity and specificity wit 95% confidence intervals. As this was a secondary analysis, an *a priori* sample size calculation was not performed.

## 3. Results

Descriptive results from the cohort have been previously reported [5]. Table 1 shows the univariate regression analysis with unadjusted odds ratios for all variables. The highest odds ratios were evident in older age groups with multiple comorbidities and prior CKD.

Table 2 shows the accuracy of various models along the patient pathway to predict ED-AKI. Models vary from 71 to 73% overall accuracy and with areas under the ROC curve from 0.765 to 0.800. Calibrations range from 14.7 to 27.4.

Table 3 presents adjusted odds ratios, raw coefficients and a simplified score for the eight variables used in model 3. The highest adjusted odds ratios are for patients with known liver disease, older age groups and diabetes. Model 3 is the preferred model because it is the simplest to use, generates the highest C-statistic, utilises information that should be available on first arrival at triage in the ED, does not depend on blood tests and has 95%CIs that overlap with model 4. Model 4 generates marginally better overall accuracy but is dependent on a point of care sodium blood test in the ED. Thus, there may be a time delay to identifying high-risk AKI patients.

Table 4 shows the predictive analyses for model 3. The most balanced probability for ED-AKI is >6 out of 31. A sensitivity of 97% can be achieved with scores <2, and a specificity of 95% can be achieved with score >13. Figure 1 shows the AUROC curve for model 3.

Table 5 (Appendix) shows the summary table for sensitivity and specificity after bootstrapping. After bootstrapping, the Youden index was 0.4878 (95%CI 0.4369 to 0.5376) at an optimal cut off at >6 (95%CI >6 to >6) giving a sensitivity of 80.5% and specificity of 68.3%.

Figure 2 shows the probability of ED-AKI based on the 31-point scoring method. At a score of 0 the probability of ED-AKI is <5%. At score <2 the probability of ED-AKI is 12%. The prediction strength of the model levels off at a score >15, where the probability of ED-AKI is 80%.

There is a positive correlation between the model 3 score and peak creatinine (r=0.415, 95%CI 0.365-0.462, p<0.0001). There was no correlation between the score and AKI stage I to III.

## 4. Discussion

In this study we have identified variables that are associated with ED-AKI and that are easily obtained in an ED. We have derived four models and identified one (Model 3) that is most practical and appropriate for the ED-triage setting. We have validated the model internally using bootstrapping and found it to be adequate for discrimination and to have good calibration. This is an objective model and a first step towards the development of a pragmatic test to guide early assessment.

This study evaluated 27 variables that are readily available either at ED triage or soon after a point of care blood gas assessment. It involved a complete group of consecutive AKI cases determined from a high-quality e-alert system. The control group from the same time period was too large for comparable evaluation so randomised selection has been used to select a similar and manageable cohort for assessment. The randomised selection provides a fair representation of non-AKI ED cases. It slants the proportion of the control group but provides a reasonable assessment for risk-stratification.

It may be argued that all patients who are likely to have ED-AKI will receive an early blood test anyway. In this case a working prediction model is interesting but unnecessary. In our clinical experience less than 30% adults attending our ED had a creatinine test. As a result, AKI may be missed and even when discovered may go undertreated. **It is currently unclear to what extent over-investigation or under-investigation of renal blood tests occur in the ED. Both our previous data [Holmes 2016] and the NCEPOD report suggest that over 40% cases of AKI are missed early and a proportion are likely to fall in the domain of the ED.** The diagnosis is often made late and consequently treatment is delayed. AKI also has a 30-day mortality that is higher than acute myocardial infarction, stroke or sepsis [5, 8, 14].

We have derived and compared six potential models based on the availability of data in a typical ED. The first four models using data available early in the ED pathway have similar correct classification at 70%, good calibration and adequate but not excellent discrimination. Models 1 and 2 are the simplest and most practical. However, with a little further interrogation Model 3 achieves the most optimal discrimination at triage and is our preferred model. This requires no blood tests and there is no need to review past records. As an early screening test, it is most appropriate.

For departments that have point of care sodium at triage, or on-site blood gas analysis, model 4 has further advantages and better discrimination. The lower level of the 95% CI for the AUROC is also greater than the AUROC for model 3 suggesting that the improvement is at least statistically significant.

These prediction models improve the objective accuracy for predicting AKI at triage. However, although they raise the probability of AKI they do not confirm it. The diagnosis still depends on requesting a creatinine test. They do, however, reduce the risk that AKI would be missed by not requesting a blood test. The early diagnosis of AKI could be markedly enhanced by point of care creatinine tests. Current practice in most EDs relies on creatinine blood samples being sent to the central hospital laboratory for analysis. This process ensures high quality results and low cost. However, there is a potential delay of at least one to two hours before a diagnosis and therefore treatment can be initiated.

One further role for predicting risk is making sure clinicians scrutinise results. Routine urea and electrolyte tests are often overlooked. Predicting risk makes sure that clinicians are more likely to chase the results of requested investigations. This in turn makes early interventions in terms of medication review, fluid balance more likely.

The diagnosis of AKI is not just a change in biochemistry. Apart from urea and creatinine blood tests the diagnosis may be made based on urine output. Whilst most patients in the ED may be evaluated for serum creatinine, few have their bladder volume or urine output measured. Urine output usually falls prior to changes in creatinine. Monitoring bladder volume and urine output could be an early warning sign for AKI and not just a late investigation for fluid balance.

Whilst this was a single-centre, retrospective study, it provides the largest cohort of ED cases so far addressing patient assessment for AKI and risk-stratification at the hospital front door and in the emergency department. It has been conducted over a five-month period and includes all AKI e-alert cases. The only other study addressing risk of AKI at the hospital front door has been conducted by the RISK investigators which provides a generalizable group assessed over a single 24-hour period with a small number of hospital AKI cases.

**Future studies should consider several important points.** The study does not include medication history, physiological observations such as a NEWS score, **physical assessment**, laboratory blood investigations, **urine output** or urine

sodium analysis all of which may yield important risk-stratification information and could improve the discrimination of the model. **However, the focus of this assessment is the triage station where lengthy and complex assessments are unwarranted. Consideration should be given to excluding certain patients who are obviously going to have a creatinine requested anyway, such as patients with critical illness.**

We have generated a model that is simple and easily accessible for every patient. The fact that it is easy to use suggests that it is more likely to be adopted into practice. The more complex the risk assessment tool and the more variables that are needed the less likely it is that it will be done at all. By the very nature of the time it takes to get all the information the object is defeated which is to get a very quick guide early on in the patients journey through A&E. An accurate medication history was not possible from the available information but should be considered in future refinements and risk-stratification models. ~~Laboratory blood tests actually realise the detection of AKI and so are not appropriate for prediction.~~

## 5. Conclusion

A risk-stratification model for ED-AKI has been derived and internally validated. This model is acceptable and objective but requires further refinement and external validation.

## 6. Disclosure of Financial Interests

The authors have no financial interest to declare and no conflict of interest disclosures.

## 7. Author Contributions

D.A.F., S.Z., T.H.R. and A.O.P. designed the study; D.A.F. and S.P. carried out the data collection; D.A.F., T.H.R. and A.O.P. analysed the data; T.H.R. made the figures; D.A.F., S.P., S.Z., T.H.R. and A.O.P. drafted and revised the paper; all authors approved the final version of the manuscript.

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## 9. Tables

**Table 1:** Univariate Cox regression analysis of prognostic factors for ED-AKI

| No | | Number of cases | Unadjusted Odds Ratio | 95% CI | AUROC | P Value |
|---|---|---|---|---|---|---|
| | **Demographic** | | | | | |
| 1 | Age, per year | 1119 | 1.038 | 1.031-1.045 | 0.706 | <0.0001* |
| 2 | Age group, per decade | | | | | |
| | Reference: 18 – 30 years | 125 | - | - | 0.708 | - |
| | >30 – 40 years | 101 | 1.105 | 0.574-2.127 | | 0.7658 |
| | >40 – 50 years | 106 | 1.438 | 0.771-2.684 | | 0.253 |
| | >50 – 60 years | 151 | 3.096 | 1.787-5.365 | | <0.0001* |
| | >60 – 70 years | 174 | 6.404 | 3.736-10.977 | | <0.0001* |
| | >70 – 80 years | 168 | 8.195 | 4.739-14.173 | | <0.0001* |
| | >80 - 90 years | 226 | 8.143 | 4.826-13.742 | | <0.0001* |
| | >90 years | 68 | 9.419 | 4.770-18.596 | | <0.0001* |
| 3 | Male Gender | 1119 | 1.417 | 1.120-1.793 | 0.543 | 0.0037* |
| | Reference: Female | | | | | |
| 4 | NH/RH Resident | 1119 | 3.502 | 2.360-5.195 | 0.565 | <0.0001* |
| | Reference: Not NH/RH Resident | | | | | |
| 5 | Previous hospital admission in 30 days | 1119 | 1.490 | 1.078-2.058 | 0.527 | 0.016* |
| | Reference: No admission | | | | | |
| 6 | Pre-admission CKD 3a-5 | 1119 | 4.446 | 3.110-6.356 | 0.598 | <0.0001* |
| | Reference: No CKD | | | | | |
| 7 | Previous recent creatinine | 1119 | 2.422 | 1.893-3.099 | 0.604 | <0.0001* |
| | Reference: No recent creatinine | | | | | |
| | | | | | | |
| | **Non-CKD Comorbidities** | | | | | |

| | | | | | | |
|---|---|---|---|---|---|---|
| 8 | Liver Disease | 1119 | 6.325 | 2.430-16.463 | 0.522 | 0.0002 |
| 9 | Peripheral Vascular Disease | 1119 | 3.932 | 1.782-8.680 | 0.519 | 0.0007* |
| 10 | Diabetes | 1119 | 3.457 | 2.487-4.805 | 0.590 | <0.0001 |
| 11 | Dementia | 1119 | 2.875 | 1.749-4.725 | 0.534 | <0.0001* |
| 12 | Lung Disease | 1119 | 2.716 | 1.829-4.034 | 0.549 | <0.0001* |
| 13 | Hypertension | 1119 | 2.570 | 2.011-3.283 | 0.612 | <0.0001 |
| 14 | Hyperlipidaemia | 1119 | 2.257 | 1.707-2.985 | 0.575 | <0.0001 |
| 15 | Active Malignancy | 1119 | 2.504 | 1.680-3.723 | 0.543 | <0.0001 |
| 16 | Cardiovascular Disease | 1119 | 1.889 | 1.453-2.455 | 0.565 | <0.0001* |
| 17 | Vasculitis | 1119 | 1.996 | 1.054-3.779 | 0.512 | 0.0297 |
| 18 | Number of comorbidities | 1119 | | | 0.712 | |
| | 6 | | 17.701 | 2.041-153.523 | | 0.0091 |
| | 5 | | 10.621 | 5.156-21.878 | | <0.0001 |
| | 4 | | 6.777 | 4.223-10.876 | | <0.0001 |
| | 3 | | 6.212 | 4.111-9.387 | | <0.0001 |
| | 2 | | 7.028 | 4.816-10.255 | | <0.0001 |
| | 1 | | 4.794 | 3.361-6.839 | | <0.0001 |
| | Reference: 0 | | | | | |
| | | | | | | |
| | ~~**ED POCT**~~ | | | | | |
| ~~19~~ | ~~Admission serum Sodium (mmol/L)~~ | ~~1119~~ | ~~0.921~~ | ~~0.898-0.943~~ | ~~0.651~~ | ~~<0.0001*~~ |
| | ~~**Investigations**~~ | | | | | |
| ~~20~~ | ~~Baseline Creatinine (umol/L)~~ | ~~1119~~ | ~~1.019~~ | ~~1.014-1.024~~ | ~~0.662~~ | ~~<0.0001*~~ |
| ~~21~~ | ~~Baseline eGFR~~ | ~~1119~~ | ~~0.977~~ | ~~0.973-0.981~~ | ~~0.698~~ | ~~<0.0001*~~ |
| ~~22~~ | ~~Admission Creatinine (umol/L)~~ | ~~1119~~ | ~~1.069~~ | ~~1.061-1.077~~ | ~~0.963~~ | ~~<0.0001*~~ |
| ~~23~~ | ~~Admission Albumin (g/L)~~ | ~~1010~~ | ~~0.876~~ | ~~0.856-0.897~~ | ~~0.726~~ | ~~<0.0001*~~ |

| | | | | | | |
|---|---|---|---|---|---|---|
| 24 | Admission Alanine transaminase (U/L) | 985 | 1.002 | 1.000-1.004 | 0.560 | 0.061* |
| 25 | Admission Bilirubin (umol/L) | 986 | 1.022 | 1.011-1.032 | 0.634 | 0.006* |
| 26 | Admission C-Reactive Protein (mg/L) | 891 | 1.013 | 1.010-1.015 | 0.763 | <0.0001* |
| 27 | Admission White Blood Cell (109/L) | 1103 | 1.108 | 1.080-1.136 | 0.641 | <0.0001* |

**Table 2:** Effect of add-on tests to C-statistics for six models for ED-AKI

| | C-statistic AUROC (95% CI) | Δ C-statistic | P Value | Hosmer & Lemeshow Test | P Value | $R^2$ | Overall Correct Classification |
|---|---|---|---|---|---|---|---|
| | | | | | | | |
| **Model 1** | 0.765 (0.740-0.790) | - | <0.0001 | 19.827 | <0.0110 | 20% | 71.05% |
| **Model 2** | 0.773 (0.747-0.797) | 0.008 | <0.0001 | 14.707 | 0.0651 | 21% | 71.13% |
| **Model 3** | 0.793 (0.774-0.822) | 0.028 | <0.0001 | 27.427 | 0.0006 | 24% | 72.21% |
| **Model 4** | 0.800 (0.776-0.824) | 0.015 | <0.0001 | 25.249 | 0.0014 | 25% | 72.74% |

**Model 1** includes data available at ED triage (N = 1119). The six variables are age, gender, nursing/residential home status, previous hospital admission, previously known CKD, and creatinine test performed in the previous 30 days.
**Model 2** includes data available at ED triage (N = 1119). The six variables are age group, gender, nursing/residential home status, previous hospital admission, previously known CKD, and creatinine test performed in the previous 30 days.
**Model 3** includes data available at ED triage (N = 1119). This model includes eight variables which are age group, gender, nursing/residential home status, previous hospital admission, previously known CKD, and creatinine test performed in the previous 30 days, and two individual comorbidities – liver disease and diabetes.
**Model 4** includes data available early in the ED patient pathway (N = 1119). The 10 variables in model 4 include nine from model 3 with the further addition of ED serum sodium available as point of care. The addition of baseline creatinine or baseline eGFR added no improvement in discrimination to the model.

$R^2$ – Cox & Snell

**Table 3: Adjusted odds ratios, raw coefficients and the score chart for Model 3 for predicting ED-AKI (N=1119)**

| Variable | Adjusted Odds ratio | Raw Coefficient | Score* |
|---|---|---|---|
| | | | |
| **Liver disease** | 7.43 | 2.01 | /8 |
| **Age Group** | | | /7 |
| **Age Group > 90 years** | 5.95 | 1.78 | 7 |
| **Age Group > 80 – 90 years** | 5.03 | 1.62 | 6 |
| **Age Group > 70 – 80 years** | 4.95 | 1.60 | 6 |
| **Age Group > 60 – 70 years** | 3.86 | 1.35 | 5 |
| **Age Group > 50 – 60 years** | 2.10 | 0.74 | 3 |
| **Diabetes** | 2.51 | 0.92 | /4 |
| **Nursing/Residential Home** | 2.14 | 0.76 | /3 |
| **Known CKD** | 2.05 | 0.72 | /3 |
| **Creatinine within 30 days** | 2.01 | 0.70 | /3 |
| **Previous hospital admission** | 1.67 | 0.51 | /2 |
| **Male Gender** | 1.44 | 0.36 | /1 |
| **Maximum** | | 7.76 | 31 |

*Score developed from raw coefficient rounded to nearest 0.25.

**Table 4: Predictive analyses for Model 3 for ED-AKI (N=1119)**

| | Sensitivity (95%CI) | Specificity (95%CI) | Positive likelihood ratio (95%CI) | Negative likelihood ratio (95%CI) |
|---|---|---|---|---|
| **To Rule OUT AKI** | | | | |
| Score <2 | 97 (95 – 99) | 33 (29 – 37) | 1.5 (1.4 – 1.5) | 0.09 (0.05 – 0.1) |
| **Balanced probability of AKI** | | | | |
| Score >6 | 80 (77 – 84) | 68 (64 – 72) | 2.5 (2.2 – 2.9) | 0.29 (0.2 – 0.3) |
| **To Rule IN AKI** | | | | |
| Score >13 | 17 (14 – 21) | 95 (93 – 97) | 3.8 (2.5 – 5.7) | 0.87 (0.8 – 0.9) |

## 10. Figure Legends

**Figure 1.**
**AUROC Curve for Model 3 Score**

**Figure 2**
**Probability of AKI based on Score from Model 3.** This shows a least squares regression curve based on the probability of ED-AKI at each score from 0 to 19 in the 31-score model. There were very few patients with scores above 19 so these have been grouped into score '19'. The trend was not linear so a least squares regression curve was chosen to present the data. $R^2$ 0.93. The coefficient for the slope is 4.4 (95%CI 3.5 to 5.3; $p<0.0001$)

## 11. Figures

**Figure 1**

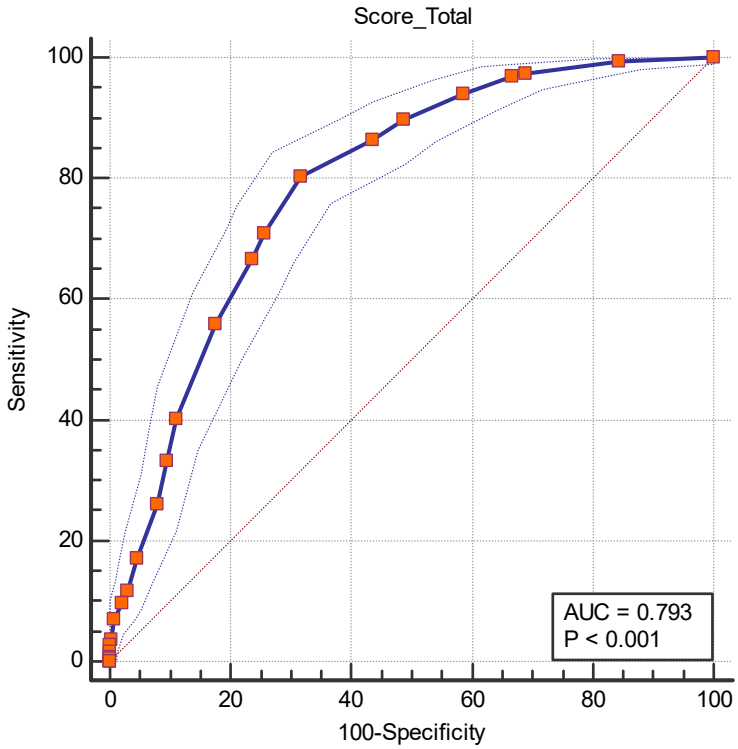
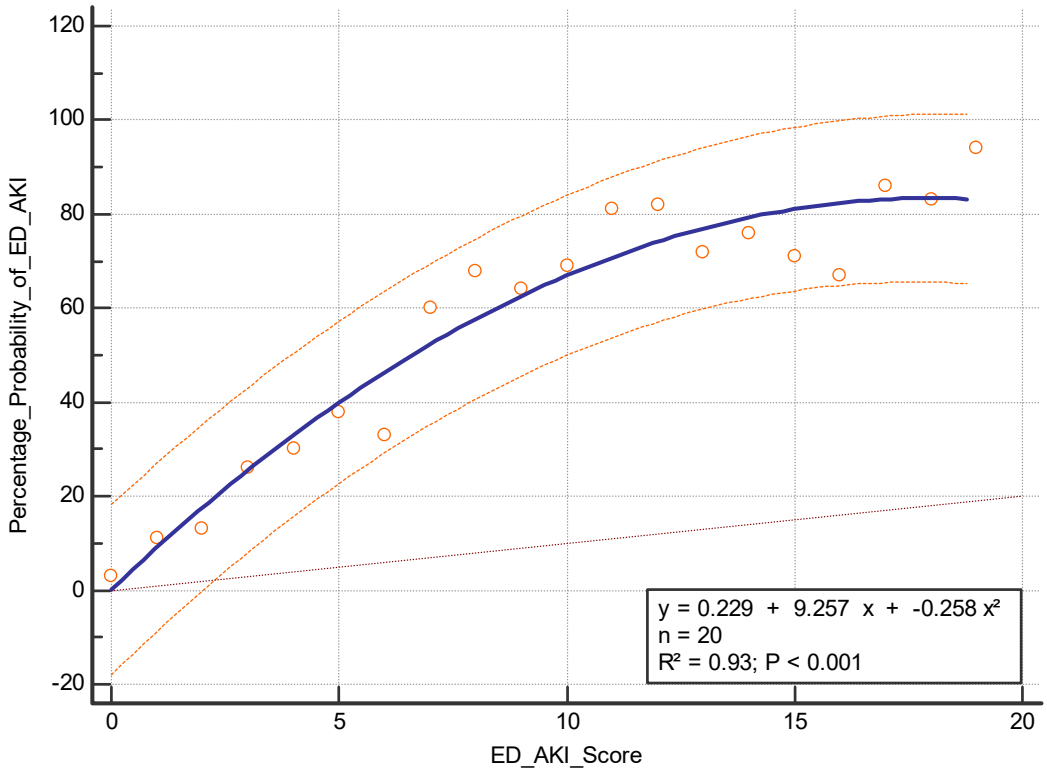


**Figure 2**




Coefficient of Determination R2 = 0.85
F-ratio=102.2; P < 0.0001

**Appendix**

**Table 5: Summary Table for estimated sensitivity and specificity for Model 3 for ED-AKI (N=1119)**

| **Estimated specificity at fixed sensitivity** | | | |
|---|---|---|---|
| **Sensitivity** | **Specificity** | **95% CI a** | **Criterion** |
| 80.00 | 68.61 | 63.19 to 73.33 | >6.05 |
| 90.00 | 50.65 | 44.58 to 56.39 | >3.95 |
| 95.00 | 39.01 | 32.55 to 44.38 | >2.71 |
| 97.50 | 30.58 | 22.51 to 36.88 | >0.97 |
| 99.00 | 19.06 | 13.31 to 25.17 | >0.22 |
| **Estimated sensitivity at fixed specificity** | | | |
| **Specificity** | **Sensitivity** | **95% CI a** | **Criterion** |
| 80.00 | 60.09 | 52.44 to 65.79 | >8.61 |
| 90.00 | 35.78 | 24.73 to 44.39 | >10.66 |
| 95.00 | 18.35 | 12.39 to 23.92 | >12.87 |
| 97.50 | 10.67 | 0.00 to 0.00 | >14.55 |
| 99.00 | 7.38 | 0.00 to 0.00 | >15.89 |

[a] $BC_a$ bootstrap confidence interval (1000 iterations; random number seed: 978).